Management of Total Gastric Necrosis Following Lye Ingestion: *

The Use of Colon to Replace Both Esophagus and Stomach

WILLIAM GRYBOSKI,** M.D., ROBERT PAGE, M.D.,
BENJAMIN F. RUSH, JR., M.D.

From the Surgical Service of the University of Kentucky School of Medicine and the University of Kentucky Hospital, Lexington

THE OCCURRENCE of total gastric necrosis following ingestion of alkali has not been previously reported, although the suicidal and accidental ingestion of these substances occurs frequently. It has been commonly observed that acid solutions affect primarily the gastric mucosa, whereas alkali burns are limited to the esophagus in approximately 80 per cent of cases.[1, 3, 5] Yudin [14] refers to severe gastric burns from alkali occurring in Russia, but no specific case reports are cited.

Dr. N. K. Yong [13] of the Department of Surgery of the University of Singapore Faculty of Medicine explains that lye burns have been a common problem in Singapore, since at one time this was a favorite method of suicide there (since superseded by plunging from the newly erected skyscrapers). Occasionally at postmortem complete gastric destruction and sometimes involvement of surrounding viscera was noted. Dr. Yong is unaware of any published reports concerning these extreme injuries or of any attempts at treatment under these circumstances.

Finney *et al.*[2] in 1960 reported a case of total gastric necrosis in an 18-month-old girl following ingestion of tinning paint. Total gastrectomy was performed about 82 hours after ingestion with primary reconstruction using an esophagojejunal anastomosis. The pathogenesis of necrosis in this case was believed to be due to the precipitation of protein by the zinc chloride present in the paint. Strode and Evans [10] described a case of 85 per cent gastric necrosis following ingestion of muriatic acid requiring a subtotal gastrectomy.

Management of an alkali burn of the stomach is complicated by the fact that both stomach and esophagus are likely to be involved.[9, 13] The occurrence of total gastric necrosis following alkali ingestion and the successful management of this case by a two-stage operative procedure is reported.

Case History

A 36-year-old Caucasian housewife attempted suicide because of severe domestic problems coupled with postpartum depression. She ingested approximately one half cup of commercial "Drano" crystals *** (NaOH) mixed with half a glass of water. She rapidly developed abdominal burning pain and vomited blood-tinged material 10 minutes following ingestion. The pain continued and she sought assistance from neighbors who had the patient drink a glass containing vinegar, water, a small amount of sodium bicarbonate and a large amount of lard. Following this she vomited ap-

* Submitted for publication April 21, 1964.

** Present address: Cedar Lake Medical Center, New Britain, Connecticut.

Supported in part by the Fred Rankin Memorial Fund.

*** The active ingredient in "Drano" is sodium hydroxide, which is the only ingredient listed on the can. Unrefined caustic soda or "lye" usually contains sodium hydroxide of not less than 95% total alkali calculated as NaOH, of which not more than 3% is Na_2CO_3.

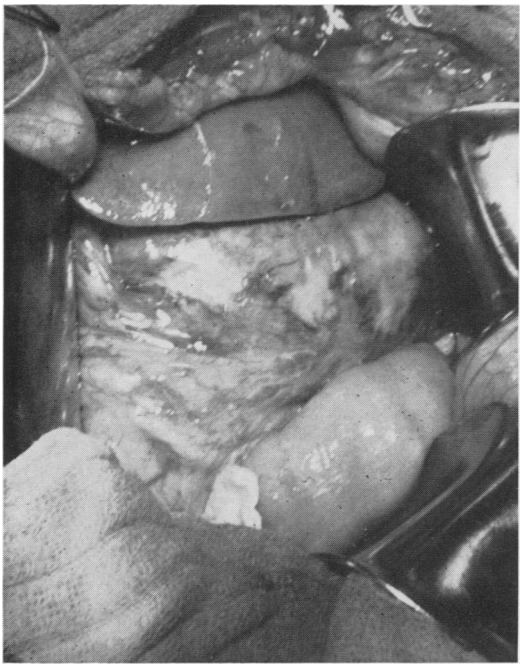

FIG. 1. Injured stomach prior to resection. Most of anterior wall is devitalized. Serpiginous dark line marks edge of huge area of full-thickness, chemical burn which extends to, but not beyond, pylorus.

proximately a quart of blood-tinged fluid. She was seen by her local physician who referred her to the University of Kentucky Medical Center for definitive treatment.

Physical examination at admission revealed an apparently healthy white female quite apprehensive and complaining of pain in her chest and upper abdomen. Vital signs were normal except for a temperature of 38.1° C. Examination of the patient's skin revealed punctate areas of alkali burn over her right cheek, chin and arms. Her tongue, buccal mucosa, palate, oral and hypopharynx were erythematous, edematous and covered by areas of patchy exudate representing alkali corrosion. The lungs had expiratory bronchi which cleared on coughing. The abdomen was soft. There was tenderness to palpation in the right upper quadrant and midepigastric area. No bowel sounds were audible.

Roentgenograms of the abdomen revealed no evidence of free air. Laboratory data indicated a hematocrit of 43% and a white blood cell count of 16,000. The patient was given codeine for pain, intravenous fluids, and oral milk hourly. Approximately 6 hours following admission her general condition began to deteriorate. She developed progressive abdominal and midchest pain. Her temperature rose to 39.4° C. She developed tachycardia and moderate generalized abdominal pain more marked in the upper quadrants. The abdominal muscles were still soft and there was no rebound tenderness. A chest x-ray film at this time demonstrated infiltration of the right lower lobe with no evidence of esophageal rupture. Electrocardiogram was normal with no evidence of pericarditis. It was believed initially that fever and pain represented aspiration pneumonitis and chemical mediastinitis.

The increasing severity of abdominal symptoms over the ensuing 28 hours with final development of definite rebound tenderness prompted exploratory laparotomy. At operation the abdominal cavity contained 200 cc. of clear, straw-colored fluid. The stomach was almost completely devitalized and fixed by its alkaline contents. The serosal surface was a mottled aquamarine color bearing large areas of necrosis (Fig. 1). The greater and lesser omentum was indurated and edematous. The majority of arteries and veins adjacent to the stomach were filled with clotted blood. As these vessels were clamped and transected, clots were squeezed comedo-like from the lumens. No definite gastric perforation was found. The duodenum was not involved. A total gastrectomy and end cutaneous duodenostomy was performed and a large sump drain was placed at the opening of the distal esophagus below the diaphragm and brought out through a left upper quadrant stab wound. At the end of the abdominal procedure cervical esophagostomy and tracheostomy were performed. The esophageal mucosa at this level was white and friable, presumably from the chemical burn. The distal stump of cervical esophagus was closed and allowed to retract into the thoracic mediastinum.

The patient was given 20 million units of crystalline penicillin intravenously. Inadvertently the drug was given much more rapidly than intended and brought about spontaneous asystolic cardiac arrest due to the sudden high level of serum potassium. This arrest was immediately noted on the monitoring cardioscope and closed chest massage was instituted. Intracardiac bicarbonate and epinephrine were given and intravenous infusion of 10% glucose and 20 units of insulin was begun to reduce the level of serum potassium. After 25 minutes of closed chest cardiac massage and the third external defibrillation, the patient spontaneously regained normal sinus rhythm. Recovery from anesthesia was normal and the patient was sent to the intensive care unit awake and alert. On the morning following operation, she had a normal electrocardiogram.

Citing the pathologist's report, histologic sections through the proximal and distal (Fig. 2A, B)

portions of the stomach "show a rather fantastic appearance of almost total gangrene of the gastric wall. The mucosa is totally destroyed. The submucosa and inner half of the muscularis is completely necrotic and there are large accumulations of neutrophils scattered between these masses of necrotic tissue. There is a great deal of acute inflammation of the surviving serosa and external half of the musculature. There are large extravasations of blood in focal areas of the submucosa—thrombosed vessels are found in portions of the gastric wall. The portion of tissue submitted as pylorus is also totally necrotic. The only viable mucosa that is encountered is at the upper end of the specimen near the area which was thought to be an area of erosion in the gross."

The patient was given intravenous fluids and large doses of penicillin and streptomycin. On the fifth postoperative day a feeding tube was placed in the duodenum. Blenderized tube feedings were given averaging 3,000 cc. (1.25 calories/cc.) each 24 hours. She remained in good electrolyte balance but over the first 3 postoperative weeks had a weight loss of 12 lbs.

On the twentieth postoperative day the second stage operation was accomplished. The procedure consisted of an esophagoduodenal interposition through a substernal tunnel with the establishment of an esophago-ileostomy, a coloduodenostomy and an ileocolostomy (Fig. 3).

Three days following the second procedure the patient developed an esophago-cutaneous fistula at the site of the esophago-ileostomy. Injection of radiopaque media revealed a stricture at the site of the esophago-ileal anastomosis and esophageal bougienage was performed. The fistula closed in 8 days and the patient was given a soft diet. She was discharged 1 month after the second operation. At this time she had gained 10 lbs. A moderate dumping syndrome was present but was well controlled by multiple small feedings of a high protein, low carbohydrate diet.

The patient returned to the outpatient department for biweekly esophageal dilatations and appeared to be doing very well.

Outpatient visits continued for 2 months. She then cancelled an appointment saying that she felt "poorly" and did not wish to come for dilata-

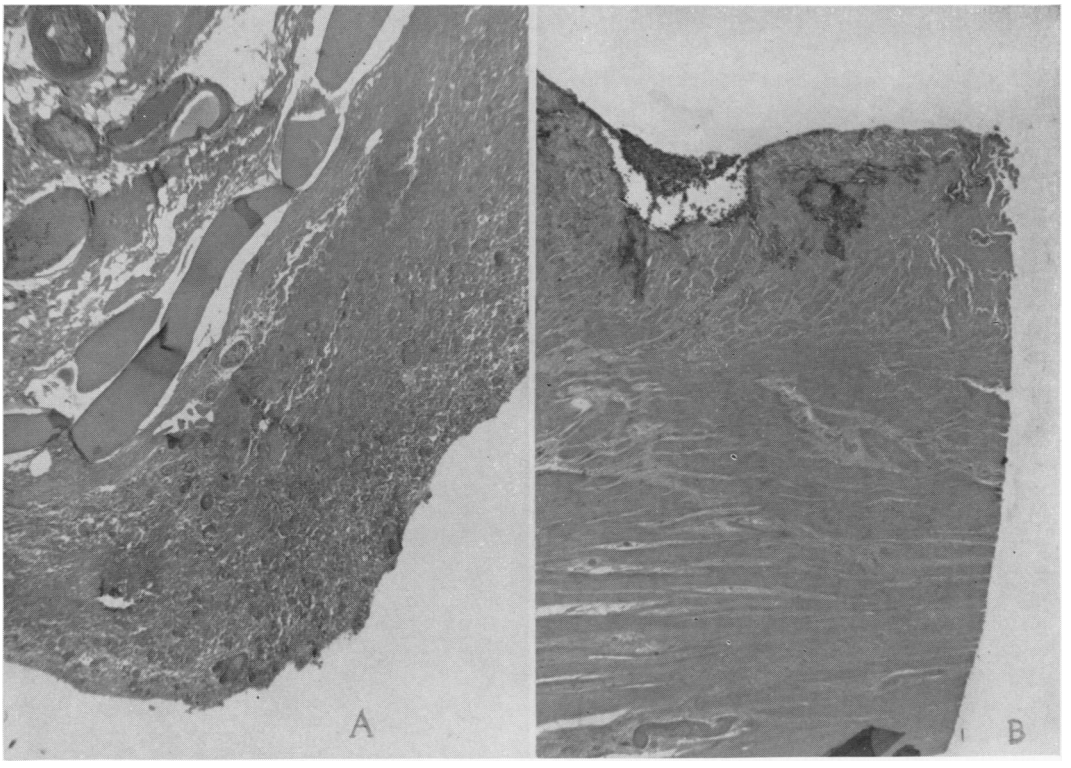

FIG. 2. A. Portion of distal stomach showing complete destruction of wall. Mucosa and muscularis are necrotic. Clots are seen in lumens of vessels. B. Destroyed wall of proximal stomach ulceration of mucosa is seen. Muscularis is a hyline homogenous mass. Most of the nuclei of the cells have vanished.

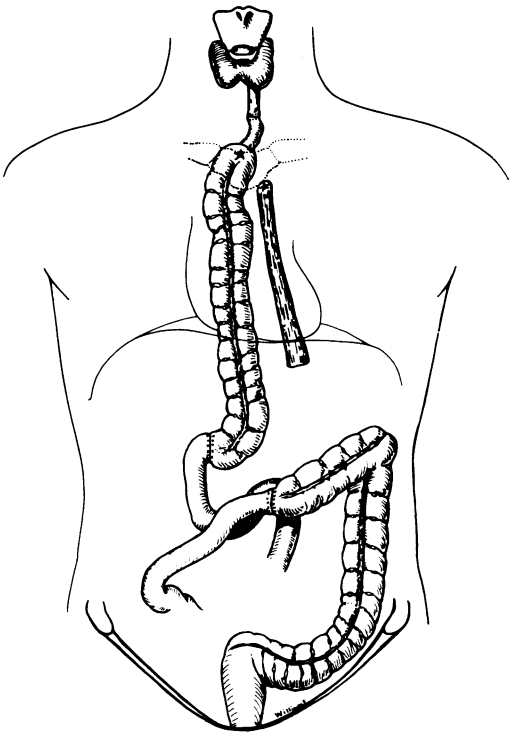

FIG. 3. Right colon has been transposed between cervical esophagus and duodenum. Intestinal continuity was restored by an iliotransverse colostomy. The necrotic and scared esophagus has been left *in situ*.

tion. Two weeks later she appeared at the hospital intensely jaundiced and profoundly ill. She was admitted at once but died suddenly 48 hours after admission.

Postmortem examination showed far advanced hepatitis with almost total hepatic destruction. On histologic examination necrotic changes, characteristic of severe serum hepatitis, were seen. This was the immediate cause of death. All suture lines of the reconstruction operation were well healed. The esophagus was thin and atretic in its upper half and slightly dilated in the lower half. It was encased in a sheath of dense, tough, fibrous tissue throughout and firmly adherent to the trachea, great vessels, pericardium and other mediastinal structures. Ninety per cent of the mucosa was destroyed and replaced by granulation tissue. A surprising finding was an old bronchial-esophageal fistula tract between the right mainstem bronchus and the esophageal remnant. This was lined with respiratory epithelium and surrounded by mature fibrous tissue. The pathologists believed that this dated to, and was the result of, the initial lye ingestion with penetration of lye from the esophagus into the bronchus (Fig. 4).

Discussion

The most common major complication of lye ingestion is esophageal stricture. Injury to the stomach by alkali occurs but is uncommon. Acid ingestion on the other hand may burn both esophagus and stomach but the esophagus often receives a lesser injury or paradoxically may be spared. Grey and Holmes [3] reviewed the literature in 1948 and found 139 cases in which swallowing corrosives, either acid or alkali, caused pyloric stenosis as a sequel. Only 19 of these strictures were due to alkali. They noted that 20 per cent of patients developing gastric stricture as a result of acid ingestion also had esophageal strictures; conversely, 20 per cent of patients ingesting alkali develop pyloric strictures and of those developing lye strictures of the stomach 70 per cent had concomitant esophageal stricture.

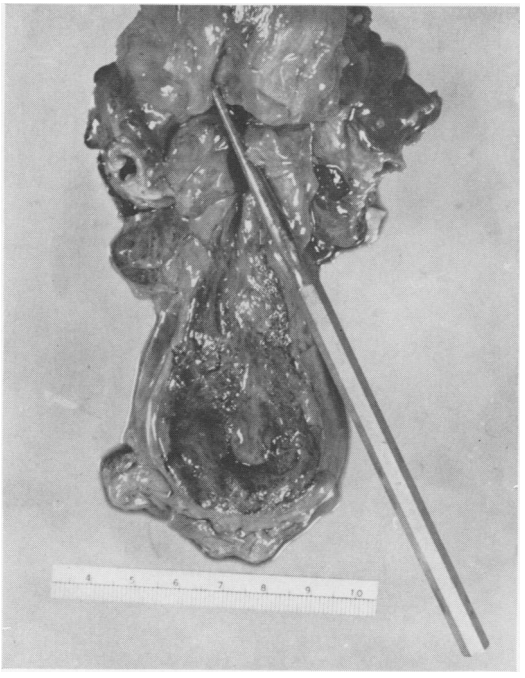

FIG. 4. Remaining esophagus laid open posteriorly. Fistula caused by lye burn joins esophagus and left main stem bronchus. Remaining lower esophagus is lined with redundant granulation tissue. The open lower end measures 4.5 cm. Upper two thirds of esophagus is almost effaced by the dense, fibrous tissue about it.

The rarity of gastric injury from lye ingestion is usually attributed to the high gastric acidity which acts as a natural antidote to the swallowed caustics. It may be that the occasional incident of gastric injury from ingestion of lye is associated with pre-existing hypoacidity or anacidity.

While we are unaware of previous reports of total gastric destruction from lye ingestion, we know of an unreported case from another institution in which the stomach was totally destroyed and at autopsy the esophagus was also completely necrotic. This knowledge influenced the management of the present patient in that we elected to abandon the thoracic esophagus. We felt that the patient was too ill to risk resection at the first operation and that the esophagus was too damaged to attempt to use it as a conduit at the second procedure. We assumed that necrosis of the mucosa and sparse esophageal glands was so complete that the risk of subsequent cyst formation in this blind remnant was slight.

The unfortunate death of this patient, 3½ months after injury, gave us the opportunity to evaluate this thesis. The esophagus was indeed severely damaged although small amounts of epithelium did remain. Whether cyst formation would have occurred in this blind segment could not be determined since establishment of a fistula between the esophagus and the bronchial tree by the initial injury provided decompression. It is worth noting that this tracheo-esophageal fistula would have been lethal in itself if the esophagus had not been promptly bypassed.

The use of right or left colon as an esophageal substitute is well established, and it has been previously used on patients with lye strictures of the esophagus.[4, 6, 7, 8] However we have not found a previous example in which the interposition was constructed between the duodenum and the cervical esophagus replacing both stomach and esophagus as in this report. This was the logical choice for this patient. The colon acts efficiently as a conduit, but not very satisfactorily as a reservoir judging by the patient's dumping syndrome.

Summary

The management of total gastric necrosis after alkali ingestion is presented. Treatment consisted of primary total gastric resection with cutaneous duodenostomy and cervical esophagostomy. At the second stage continuity was re-established between duodenum and cervical esophagus by interposition of a segment of colon. To our knowledge such a complication from ingested lye and this type of colon interposition have not been previously reported.

References

1. Arena, J. M.: Pyloric Stricture following the Ingestion of Muriatic Acid. South. Med. J., **29**:331, 1936.
2. Finney, D. C. W., C. Schnaufer and E. S. Stafford: Total Gastrectomy in an Infant Made Necessary by Ingestion of Tinning Paint. Ann. Surg., **151**:891, June, 1960.
3. Gray, H. K. and C. L. Holmes: Pyloric Stenosis Caused by Ingestion of Corrosive Substances. Surg. Clin. N. Amer., **28**:1041, 1948.
4. Groves, L. K.: Colon Replacement of the Esophagus. Amer. J. Gastroenterol., **38**:171, 1962.
5. McLanahan, S.: Pyloric Occlusion Following Ingestion of Corrosive Liquids. J.A.M.A., **102**:735, 1934.
6. Nardi, G. L.: Surgical Treatment of Lye Strictures of the Esophagus by Mediastinal Colon Transplant without Resection. New Engl. J. Med., **256**:777, 1957.
7. Reynolds, J. T., V. L. Guynn, W. J. Grove and H. Javid: Experiences with Right Colon Replacement of Esophagus. Amer. J. Surg., **101**:39, 1961.
8. Scanlon, E. F. and C. J. Staley: The Use of the Ascending and Right Half of the Transverse Colon in Esophagoplasty. Surg. Gynec. & Obstet., **107**:99, 1958.
9. Schulenburg, C. A. R. and M. B. Cape Town: Corrosive Stricture of the Stomach without Involvement of the Esophagus. Lancet, **241**: 367, 1941.
10. Strode, E. C. and M. L. Dean: Acid Burns of the Stomach—Report of Two Cases. Ann. Surg., **131**:801, 1950.
11. Tucker, A. S. and E. W. Gerrish: Hydrochloric Acid Burns of the Stomach. J.A.M.A., **174**:890, 1960.
12. Vinson, P. P. and H. R. Hartman: Pyloric Obstruction Due to Swallowing a Solution of Concentrated Lye. Med. Clin. N. Amer., **8**: 1037, 1925.
13. Yong, N. K.: Personal communication.
14. Yudin, S. S.: The Surgical Construction of 80 Cases of Artificial Esophagus. Surg. Gynec. & Obstet., **78**:561, 1944.